MOORE, (W. O.)

OCULAR SYMPTOMS IN DISEASES OF THE SPINAL CORD.*

By WILLIAM OLIVER MOORE, M.D.

PROFESSOR OF THE DISEASES OF THE EYE AND EAR IN THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL, ETC., ETC.

OF Nature's minute wonders the human eye is the paragon. But it is not the apparatus which the delicate knife of the anatomist reveals, the retina and lenses, or even their combined arrangement, that most strikingly indicates the subtile workmanship involved in the little fleshy globule we call the eye; it is the effects they produce, the purposes they subserve, the results they accomplish. Far greater are these than the careless crowd dream of, or the imaginative fully realize. The phenomena of sight is indeed sufficiently extraordinary; not less so are the minor missions which the visual organ fulfils. The eye speaks with an eloquence and truthfulness surpassing speech. It is the window out of which the winged thoughts often fly unwittingly. It is the tiny magic mirror on whose crystal surface the moods of feeling fitfully play, like the sunlight and shadow on a stream. How aptly has the eye been called the "window of the soul"; instinctively it is raised in devotion, and bent downward in shame. When enthusiasm lends fire to the soul the eye flashes; when pleasure stirs the heart the eye sparkles: when deep sorrow darkens the bosom the eye dispels hot tears; when confidence stays the mind, the eye looks forth proudly; when insanity desolates the brain, the eye roves wildly; and

> "O'er the eye Death most exerts his might,
> And hurls the spirit from its throne of light."

---

* Read before the Section on Neurology, N. Y. Academy of Medicine, March 9, 1888.

SURGEON GEN'L'S OFFICE 338 LIBRARY

Eye language is thus a part of our study in medicine, and should be more generally understood than it is, not in diseases of the brain and cord, but in general affections. The unfortunate know a friend and are reassured ; the timid recognize a master spirit and are strengthened ; the guilty know their accuser, and quail. Beware of the man whose eye you can never meet.

That the eye should show changes in the diseases affecting the brain, with which it is so closely connected, is clear, but why, in those situated in the spinal cord, it is not quite so easy to comprehend. Ever since the diseases of the "golden bowl" have been the subject of close study, ocular symptoms have been noticed, and of late years these same symptoms, associated with affections of the "silver cord," have claimed much attention and enquiry.

I regret that I am unable to make any new contribution to our knowledge of the pathological anatomy of these diseases, but hope that in the discussion it may be evoked.

In entering this vast and interesting field one must be very careful to make minute and accurate observations, especially in reference to pupillary signs, as Iwano has found that out of 134 young persons examined, 131 had assymetry of the face, and that the pupils were irregular in 122 : and that the largest pupil occurred most frequently on the left side, which was usually the smaller side of the face.

We shall not attempt to cover the above field, which the title of the paper indicates, but only take up certain points that have always interested us.

Your attention will be more particularly called to atrophy of the optic nerve, and ophthalmoplegia interna, with but only a passing notice to ophthalmoplegia externa, and nystagmus.

Atrophy of the optic nerve comes chiefly under the notice of the ophthalmologist, while the affections of the iris more commonly under the observation of the physician.

The ocular symptoms in spinal cord disease are to be looked upon as associations and not as effects ; and that they are also always the result of degenerative processes in the cord.

The usually slow progress of the spinal symptoms is evidence of this fact.

Among the many degenerative diseases of the cord it is strange that most of the eye symptoms should be with one of them alone, with tabes dorsalis. The fact that they are associations and not effects is clearly stated by Gowers in the following manner :

1. "That disease of any nature may exist in any part of the spinal cord without the occurrence of ocular symptoms, if we except the very rare paralysis of the dilators of the pupil in disease of the sympathetic tract in the cervical region.

2. "The ocular symptoms which may be absent when the cord disease is advanced may exist in extreme degree when such disease is in a very early stage.

3. "With the single exception of the sympathetic symptoms just mentioned, we know of no anatomical connection or functional mechanism by which the spinal cord disease can produce the ocular symptoms."

INJURIES TO THE SPINE.

Experimentally in laboratories, injuries to the spinal cord of animals have shown ocular symptoms, and these naturally lead us to expect similar results in man, where injuries of the spinal column have inflicted pressure on the cord.

Mr. Erichsen,* of London, has done much to bring this to notice, and all but considered it sufficiently frequent to establish a causal relation between the two events.

It is stated on authority, that Alexander the Great was in danger of losing his eyesight from the blow of a heavy stone on the back of the neck. Thirty-six per cent. of Erichsen's cases showed undoubted eye symptoms.

These are more commonly : difficulty of seeing in poor light to read, blurring of the type, floating bodies before the eyes, occasionally diplopia, with photophobia. The ophthalmoscopic appearances are usually negative.

Wharton Jones† considers the eye symptoms in these

* Erichsen, "Concussion of the Spine," London, 1875.

† "Failure of Sight after Railway and other Injuries of the Spine and Head," London, 1869.

cases due to the disturbance in the cilio-spinal centre, and the sympathetic filaments springing from the dorsal and cervical cord.

Other authors have reported severe eye symptoms from comparatively slight injuries of the spine.

I have seen but two cases of concussion of the spine, and in each of these eye symptoms were present in the shape of asthenopia. The fundus in each case was normal, and distant vision good, yet difficulty was experienced on reading; both were females, and each had loss of accommodation, that is to say, they could not use the ciliary muscle long without fatigue, and a convex glass had to be given to rectify this difficulty. In neither case was muscular disturbance of the extrinsic muscles of the eye found.

In Caries of the Vertebræ, when of traumatic origin we frequently find intra-ocular symptoms, and the ophthalmoscope usually shows a condition of engorgement of the vessels of the optic disc and surrounding parts.

Dr. Chas. S. Bull * reports the histories of eleven cases of traumatic caries of the spine, mostly occurring in the cervical, but some also in the dorsal and lumbar regions.

Seven had engorgement of retinal veins, with no changes in the arteries: in two choked disc was noticed, and in two anæmia seemed to be present, although the veins were enlarged.

I have examined fifteen cases of ocular disturbance in vertebral caries of traumatic origin, and found the following conditions: In two, optic neuritis, with swelling: three, hyperæmia of the disc, with blurred outline, but no œdema; eight had enlarged veins, and arteries normal in size or only slightly enlarged, the changes were symmetrical in each eye; two had floating bodies in the vitreous, with enlarged retinal veins; these changes are probably due to the sympathetic filaments springing from the dorsal and cervical cord.

LOCOMOTOR ATAXIA.

The frequency with which optic nerve atrophy occurs in tabes is differently stated; Leber found it present in 26 per

* Trans. American Ophthal. Society, vol. ii.

cent., Gowers, in 20 per cent., and Nettleship, in 50 per cent.

During the past ten years we have made notes of 80 cases of atrophy of the optic nerve, where no other history could be obtained, and of this number 32 had the signs of tabes as shown by the absence of the knee-jerk; 18 had ataxic symptoms when first seen. In examining the reports of many of the ophthalmic hospitals of this country, we are unable to tell the proportion of optic nerve diseases due to locomotor ataxia, owing to the method of recording diagnoses. Erb found only 12½ per cent. of his cases to have atrophy of the optic nerve; so that, although it is admitted by all writers as a frequent association, the percentage is at great variance. It has been said that the percentage given by the ophthalmologist is too high: I do not think so, but rather that it is due to the fact that the patient seeks advice for failing vision, when no signs of ataxia are yet present, and that the observers being on the alert for a cause of the optic atrophy, enter into an examination of the general condition of the patient and discover tabetic symptoms. I know this has been my personal experience in this direction. Atrophy of the optic nerve belongs to the more frequent of the complications of tabes; it commonly arises in the initial stage, and may be the first manifestation; the amblyopia produced by it may last for as many as ten years, before other symptoms of tabes appear, as in the case reported by Charcot. Gowers has also reported similar cases; in one the optic atrophy preceded the locomotor symptoms twenty and in another fifteen years. That optic atrophy ever occurs before the loss of the knee jerk, I am not satisfied; in all the cases under my observation it was absent—and I believe it is well recognized that the loss of the knee jerk may precede for a long time other locomotor symptoms.

When the disease was supposed to be situated and limited in the posterior columns of the cord, the association with it of a peripheral degeneration of the optic nerve was an anomaly. But since the recent pathological researches of Pierret, who has shown that the degeneration of the optic

nerve is not the only peripheral lesion, and that that in the cord is not the only central change. He has shown that there is often an independent degeneration in the cutaneous nerves, commencing in the extremities, and that the optic nerve change is strictly analogous ; that there may also exist a degeneration at the central termination of the optic as well as of other cranial nerves, similar to that existing in the posterior columns of the cord. From his standpoint tabes is considered a "wide sensory neurosis," in the course of which the optic nerve atrophy occurs. Déjerine and Westphal have also enlarged our former narrow conceptions of the tabetic process. During the period when there is no affection of the patient's gait, and only the loss of the patellar reflex, unsteadiness on standing with bare feet close together, with closed eyes, and with lightning pains, there is no doubt that optic atrophy often commences and advances to a considerable degree, and according to Gowers, it occurred during this period twice as frequently in this than the later stages of the affection. In my cases, 14 out of 32 had only loss of knee jerk and rheumatic pains in the legs. It is rare for it to occur when the ataxia is so great that the patient cannot walk.

The atrophy usually begins in one eye before the other, and may reach a considerable degree before the fellow-eye suffers. The immediate cause of the amblyopia in tabes is the gray degeneration of the optic nerves—a degeneration similar in all respects with the changes in the spinal cord. One disease begins at the periphery of the trunk of the optic nerve, and gradually attacks the central fibres ; it always begins on the trunk of the nerve or the portion nearest the eye, and from thence toward the optic tract. The narrowing of the visual field would lead us to this conclusion, had not post-mortem changes already shown this condition.

The disease of the optic nerve manifests itself by diminution in the acuteness of vision ; the field which is at first normal becomes gradually narrower, the field being irregularly contracted. The color field is usually limited before the visual, the perception of green being lost first, then red, and then yellow and blue. Ophthalmoscopic examination

shows in the early stages a dirty-looking nerve, which is indicative of a low grade of neuritis, and this continuing produces the final full marked appearances of white atrophy of the optic nerve, with narrow arteries and other parts of the fundus appearing normal.

The optic nerve atrophy occurring in tabes is usually progressive, and leads to complete blindness, though I have seen two cases where vision was not completely destroyed. And one may observe no ophthalmoscopic change in the fundus, and yet amblyopia of a high grade present; in such cases doubtless degeneration is retro-bulbar.

The next most frequent ocular symptom in tabes is the pupillary change. Of the four muscular actions, contraction of the pupil on stimulation of the optic nerve, contraction of the sphincter, in association with that of the ciliary and internal recti muscles, and contraction of the dilator fibres of the iris on stimulation of the skin, and contraction of the ciliary muscle on accommodation, some or all may be lost in association with spinal disease. These changes depend upon or leave three centres capable of separate action, all of which probably lie in the tract beneath the aqueduct of Sylvius. Experiments made by Hensen make it likely that the anterior portion of the tract governs accommodation, and the centre next behind it the reflex contraction of the iris. On the outer side of the latter is a centre on which depends the reflex sensory dilatation of the pupil. The efferent path of the two former are through the third nerve. As yet we know little as to the centre for the contraction of the iris which is associated with accommodation, not knowing whether the nucleus for the ciliary muscle is connected with the mechanism for contraction of the pupil at the centre, or in the lenticular ganglion, or in the ganglionic mechanism within the eye. It is more probable that the connection is in the lenticular ganglion.

The path by which stimulation of the skin causes reflex dilitation of the iris is circuitous. The afferent impulse reaches the centre by the cervical part of the spinal cord when the skin of the neck is stimulated, and the efferent impulse descends the cervical cord thence to the superior

thoracic ganglion of the sympathetic, and then ascends the sympathetic to the eye.

These pupillary symptoms are as common in tabes as they are rare in other spinal diseases.

The most common being the loss of reflex action to light, while the pupil still contracts to accommodation, the "Argyll-Robertson" pupil or reflex iridoplegia.

Associated with this symptom is also a loss of the dilitation of the pupil on stimulation of the skin. Next in frequency, but very much less common, is paralysis of all the intrinsic muscles of the eye, the ophthalmoplegia interna of Hutchinson.

The rarest is loss of accommodation, cycloplegia, without loss of reflex action.

If we embrace all of the pupillary symptoms, both slight and transitory, and the more profound, we will find them present, according to Erb, in more than one-half of all tabetic cases. Gowers, in 72 cases of primary degenerative ataxia, found the internal muscles of the eye affected in 92 per cent.

The percentage of his cases with pupillary signs was in the first stage 84, in the second 93, and in the third 100.

In forty-one of our own cases already spoken of, the "Argyll-Robertson" pupil was present.

When the light reflex is lost the pupils are often small, but not necessarily so.

When there is loss of accommodation they are rarely very small, less than 2½ mm., and are often 4 or 5 mm. in diameter.

The reflex dilatation of the pupil when the skin is stimulated is a phenomenon closely allied to the contraction of arteries, which may be produced in animals by the stimulus of pain. The dilatation of the pupil may be obtained by irritating the skin of the face or the neck; it is double, that is, the stimulation of one side causes dilatation of both pupils.

The skin reflex is usually absent when the light reflex is lost; the skin reflex may be retained when cycloplegia is present.

Dr. Hughlings Jackson has reported a case where vision was absolutely lost by optic atrophy, in whom the "Argyll-Robertson" pupil existed, and when the patient "made believe" look at the clouds the pupil enlarged, and contracted when he made the effort to look at a near object.

In examining the reflex dilatation which accompanies stimulation of the skin, precaution as follows should be observed: the eye should be shaded from a glare of light, voluntary movements on the part of the patient should be arrested during the test, as any movements being made the pupils being shaded will cause dilatation of the pupils.

Urthoff* found reflex immobility of the pupil, combined with preservation of reaction on convergence, in 67 per cent. of all tabetic cases, and also the pupils were found to be unequal in one-fourth of the cases; one-sided cycloplegia was found in only five out of 166 cases. Reflex immobility of the pupil, without reaction on convergence, was found in thirty cases.

We must not omit the paralysis of the externe muscles of the eye, that are so commonly seen in tabes, usually either the abducens or the motor oculi, and rarely the fourth nerve, which give rise to various symptoms of dizziness, diplopia and strabismus. Graefe has pointed out that tabetic patients show little disposition to fuse the images in binocular vision, and this is taken as sign of the central origin of the affection. These muscular paralyses are frequent in the early stages of the disease; they are transient often in character, and this fact has not been very readily explained. Ptosis is also present when no other branch of the third nerve is involved.

In 203 cases of tabes collected from various sources, paralysis of ocular muscles occurred in 52, or over 25 per cent.

GENERAL PARESIS OF THE INSANE.

In this disease ocular symptoms are quite prevalent and in great variety. Mr. W. B. Lewis, of Wakefield, England, has collected the histories of sixty patients, with the following result: Loss of reaction of the pupil to light, 78 per cent.;

* Berlin, "Klinisch Wochenschr." 1886, No. 3.

movements on accommodation, associated iridoplegia, 43 per cent.; reflex dilatation to cutaneous stimulation was lost in 63 per cent.; complete fixity of the pupil, without impairment of accommodation, was found in 15 per cent.; in 7 per cent. of ophthalmoplegia interna occurred, although it was rarely complete as regards the ciliary muscle.

He summarizes as follows:

1. A loss of reflex dilatation to sensory stimulation occurs in a very large proportion of cases.

2. Reflex iridoplegia (loss of action to light) next to the preceding is the most frequent accompaniment of the disease.

3. Complete loss of movements of accommodation occurred in 25 per cent.

4. Cycloplegia associated with the latter in four of the 60, (more or less complete) and was found only in the advanced stages of the disease.

And he concludes that the sequence of morbid phenomena occurring in the iris, is as follows:

Paralysis of reflex dilitation to cutaneous stimulation, reflex iridoplegia probably shown at first by an initial contraction, followed by dilitation under full focal light, and passing into a later stage of immobility, and occasionally complete, ophthalmoplegia interna. Optic nerve atrophy occurs, and in 22 cases examined by Dr. Lawford, London, Eng., three were found thus affected.

Nystagmus is associated with tabes, but is a rare symptom in this affection, and is usually found in multiple-sclerosis; it has been ably set forth by Friedreich, who states that it is always bi-lateral, and that the movements of the eyes are rotatory and irregular, as seen in the case presented to the Society this evening:

Richard Purcell, æt. 42, watchman. Sent to me by Dr. A. C. Coombs, of Newtown, N. Y., Feb. 13, with the following history: That for the past fourteen months he has noticed a difficulty with his eyes; that they "jump" and annoy him very much by causing objects to move rapidly in front of him. Has no pain. In 1872 he was struck on the left shoulder by a falling telegraph pole; this knocked him over

and caused a severe contusion of the arm and shoulder, so that he could not use it for many weeks, and he never has had freedom from pain in it since, although he has moderate use of the member. Has not used tobacco since 1886, but has used alcohol in moderation.

Present condition, Feb. 13, 1888, well nourished and all the functions good. R.=$\frac{15}{18}$, L.=$\frac{15}{30}$—2.5 D, ◯—2.75 D, cy. ax. 30°. Abduction, 8°; adduction, 15°; no vertical deviation by the prism test, nor hyperphoria at 20 feet. With the correcting glasses in position the nystagmus is the same as without them. There is no contraction of the visual or color field as tested by the perimeter.

Both eyes show a marked nystagmus both around a transverse as well as an antero-posterior axis, which gives the eye a peculiar, swimming appearance. When the patient looks downward the ocular movements cease, as they also do when he fixes the eyes on a given object, but as soon as they are removed the oscillations begin with marked vigor.

Pressure on the nape of the neck by the fingers, or throwing the head back so that the neck impinges upon the collar, causes the motions of the eye to cease. When lying in bed the patient states the eyes are quiet.

The fundus in each eye shows a dirty-looking optic nerve, but one which would not be considered abnormal by many; yet I am inclined to consider the nerve in a state of neuritis of a low grade which will produce atrophy; the ophthalmoscopic examination is very difficult, owing to the rapid movements. The pupil responds to light and accommodation.

The knee-jerk is present so far as my testing has proved; the patient complains of swaying while walking, and that his feet do not have the proper sensation on touching the floor. I present him as an example of nystagmus probably due to multiple sclerosis, from the injury of the shoulder years ago.

It does not occur during rest, but always on an attempt to fix the eye.

Frederich considers it due to a form of ataxy of the movements of the eye, and speaks of it as ataxic nystagmus, and the

cause to be due to a disturbance of the co-ordinatory tracts, which lead from the centres of co-ordination to the nuclei of the nerves of the ocular muscles lying on the floor of the fourth ventricle, and that it does not occur in spinal disease until the medulla is involved.

Pierret explains these movements on the ground of the primary disease of the sensitive root tracts of the trigeminus in the medulla.

Nystagmus is a very common symptom of multiple sclerosis, and it is increased by any effort of the will or any violent emotion ; according to Charcot it is met with in one-half of the cases.

In multiple sclerosis of the cord, we have temporarily a permanent diplopia due to paralysis of the various ocular muscles ; amblyopia is also observed, but it rarely, as in tabes, leads to positive blindness, with optic nerve atrophy, although in rare cases it does ; the difficulty is probably due to nodules of sclerosis in the optic tracts and optic nerve.

The paralyses of the external muscles of the eye due to spinal cord disease, we will reserve mention of till a future occasion, as the subject of ophthalmoplegia externa is of much scope and interest.